

CAPSULE



apsule

Together to cross the 1st pathway

**SECOND YEAR
PATHOLOGY
REVISION 1**

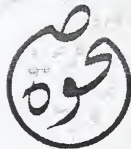
DR . NADIA GALAL (CELL INJURY)



CAPSULE

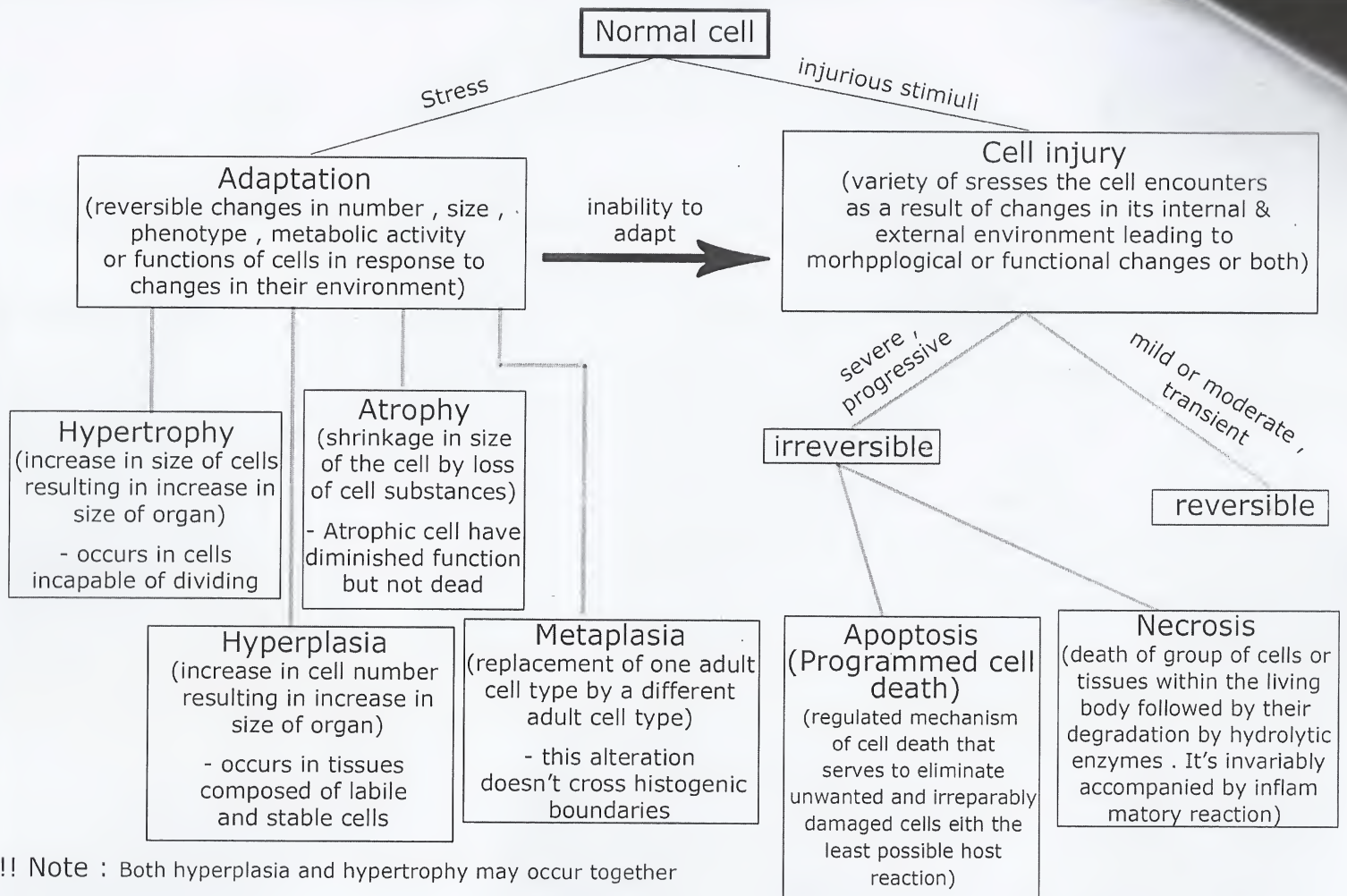


CAPSULE TEAM



SAHWA FAMILY

CAPSULE



-: Adaptation :-

Hypertrophy

* Physiologic

- ① Uterine smooth muscle in pregnancy (accompanied by hyperplasia)
- ② Skeletal muscles in athletes.

* Pathologic

- Left ventricular in systemic hypertension and aortic valve disease.

Hyperplasia

* Physiologic

- ① Hormonal
 - a Female breast
 - b Pregnant uterus
- ② Compensatory
 - a Regeneration of Liver after hepatectomy
 - b Following nephrectomy on one side

* Pathologic

- ① Hormonal
 - a Fibrocystic disease of breast (estrogen excess)
 - b endometrial (estrogen excess)
 - c Thyroid (Goiter) (TSH excess)
 - d prostatic
 - e Bone marrow (erythropoietin excess)
- ② Epithelial due to chronic irritation
 - Brun's nests in bilharzial cystitis
- ③ Reactive lymphoid in case of infection

Atrophy

* Physiologic

- atrophy of brain and heart with aging.

* Pathologic

- ① disuse atrophy
- ② Neuropathic (denervation)
- ③ endocrine: Loss of its stimulation
- ④ Pressure atrophy
- ⑤ inadequate nutrition

Metaplasia

A Epithelial

- ① Squamous
 - From Pseudo-stratified Columnar ciliated epithelium (in bronchi, in nasal polypi and rhino scleroma)
 - From Simple Columnar epith. (endocervix, gall bladder)
- ② Columnar
 - in Barrett's esophagus

B Mesenchymal

- Osseous
- Cartilaginous
- Myeloid

Notes

- ① In Atrophy → there is ↓ Protein synthesis, ↑ Protein degradation.
- ② Metaplasia is often response to chronic irritation to withstand stress.

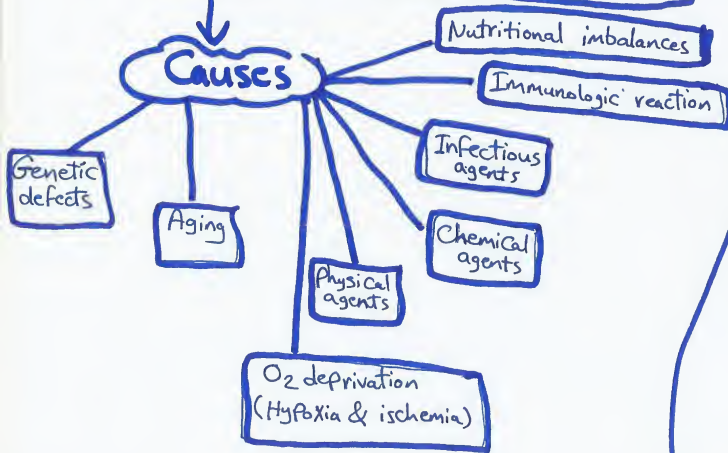
Cell injury

Note:

although there are no definitive morphological or biochemical correlates of irreversibility but there are 2 phenomena characterize

Mitochondrial dysfunction
disturbance of mem. function

Causes



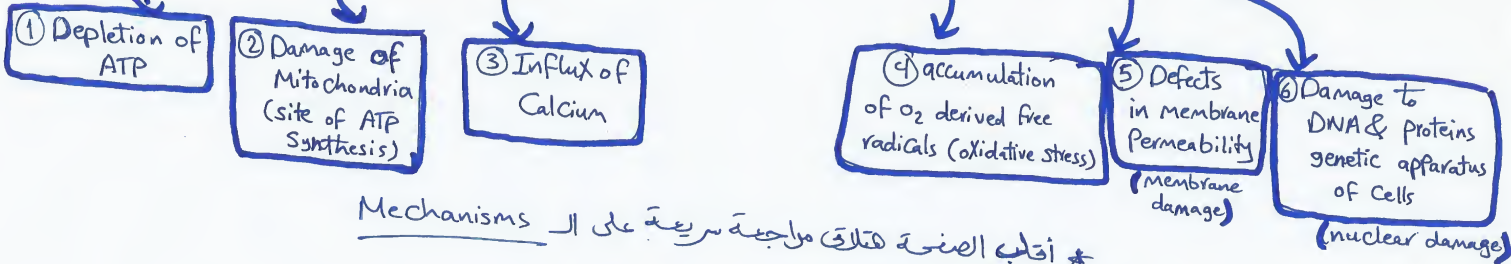
Pathogenesis

A) The type of injury, its duration and its severity

B) The Type, status, adaptability and genetic Makeup of Cell injury

* Striated muscle (skeletal muscle) withstands ischemia for 2-3 hours but Cardiac muscle dies after only 20-30 minutes.

Mechanism



* أقالب الصنة هتلاق مراجعة سريعة على الـ Mechanisms

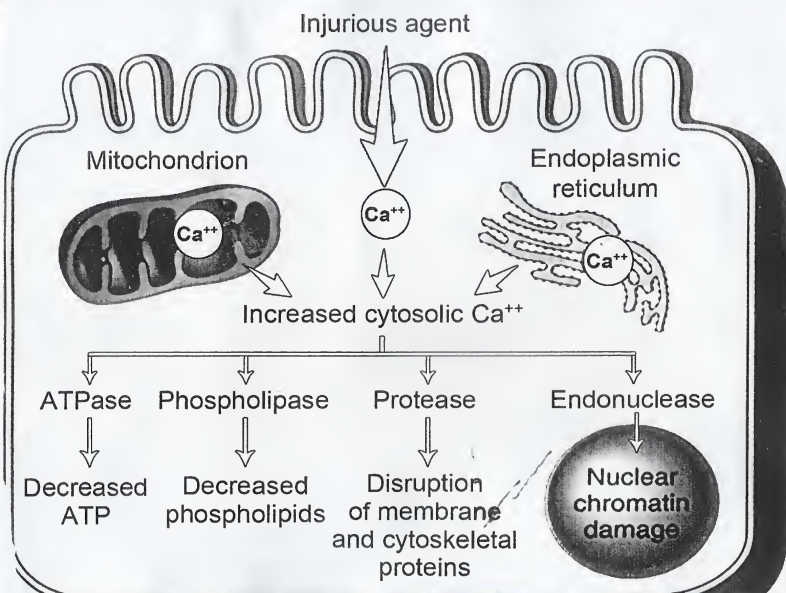


Fig. 2.10: Effects of increased cytosolic calcium in cell injury (Influx of Ca^{++})

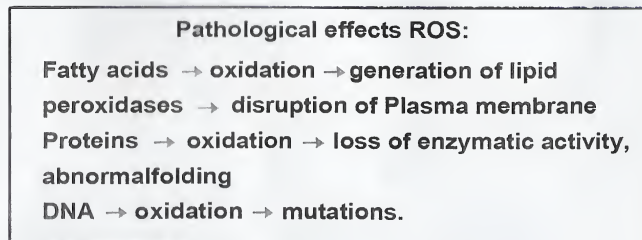


Fig. 2.11: Pathological effects of accumulation of oxygen-derived free radicals

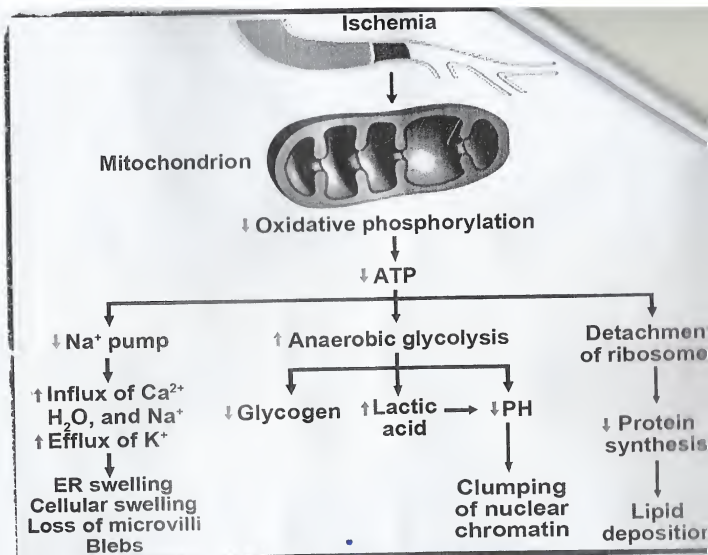
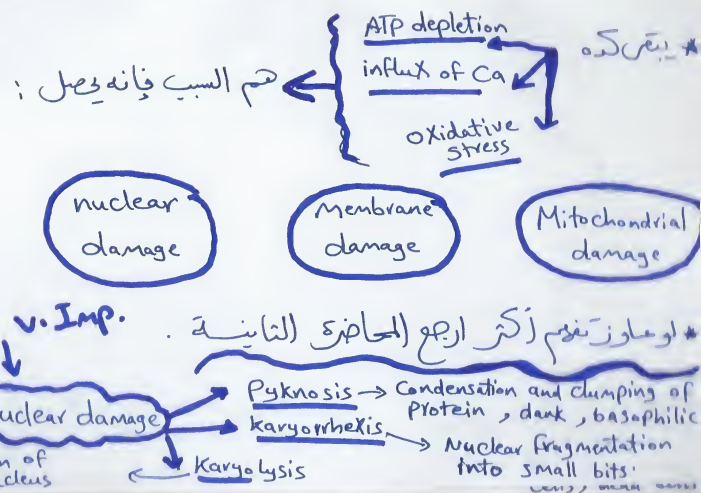


Fig. 2.9, Effects of ATP depletion



Types of Cell injury

Reversible

Morphology

A) Hydropic change

→ accumulation of water

→ Pathogenesis: Impaired regulation of Na & K

↓
Na الـ يدخل الخلية ويدخل
ماء الـ water

→ Grossly: organ → Pale → ↑ in weight

→ Microscopically → Small clear vacuoles within cytoplasm.

Fibrinoid

→ Ex: Vasculitis, Polyarteritis nodosa, Malignant hypertension.
→ Grossly: bright pink & amorphous
→ Micro: eosinophilic, hyaline

C) Mucoïd change

B) Hyaline change

Irreversible

Necrosis

Def. → في أول ورقة في البراقعة والتعريف ثم فن.

Morphology: More eosinophilic, nuclear changes

Types ← (فيه ٣ أنواع: الخافقة، التالفة، الموت)

Apoptosis

Karyolysis
Karyorrhexis
Pyknosis

Coagulative

→ Ex: infarction heart, spleen, kidney

→ grossly: pale yellow, swollen, firm, opaque

→ Microscopically: Dead cells with preserved basic tissue architecture but details (nuclei) are lost.

Liquefactive

→ Ex: infection in brain, pyogen abscess, Amoebic abs.

→ grossly: turbid fluid, leaves cavity (cyst)

→ Micro: Creamy white pus, there's cyst

Caseous

→ Ex: TB

→ grossly: friable yellow white, look like cheese or casein of milk

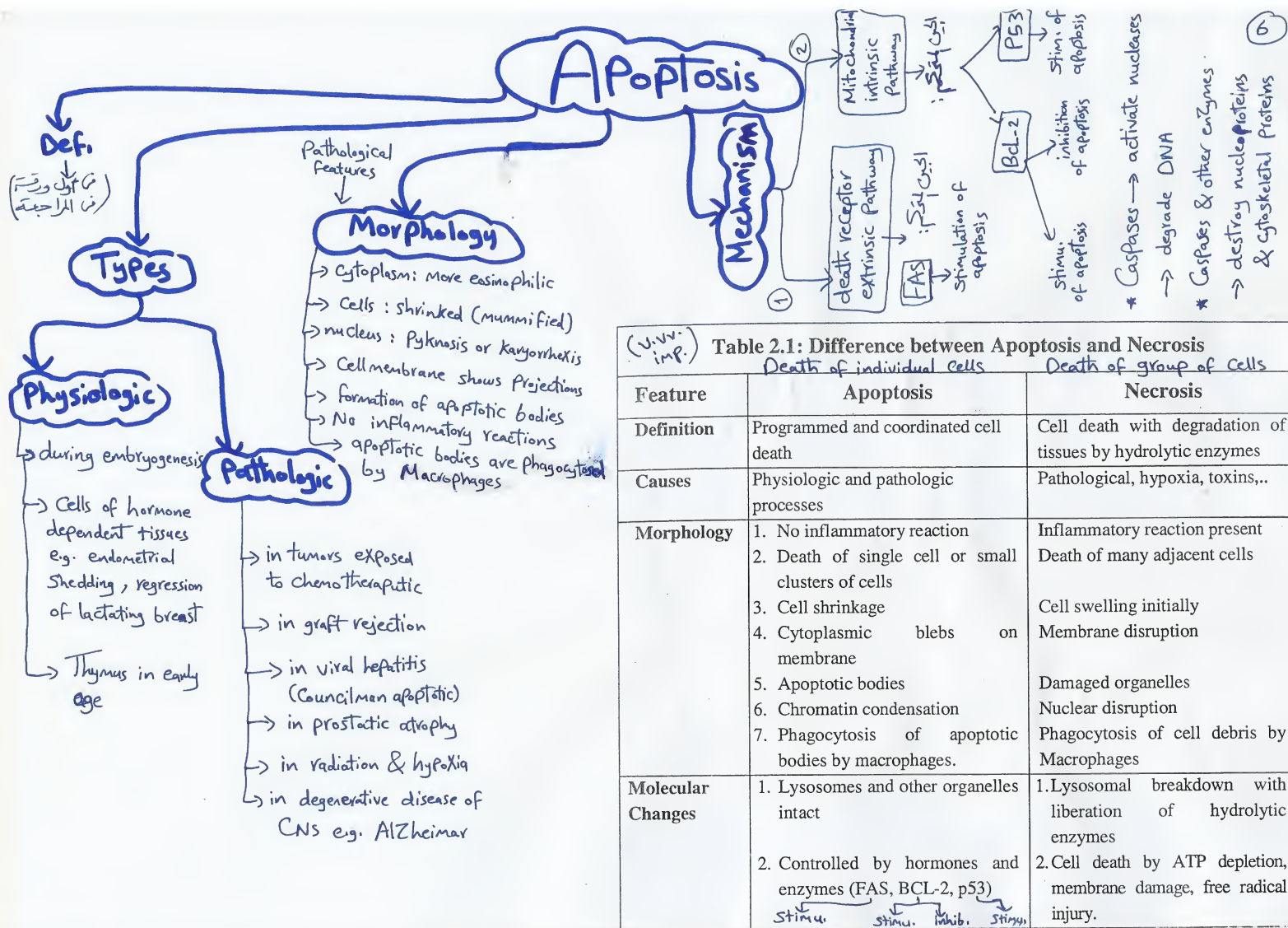
→ Micro: Complete loss of tissue architecture

Fat

→ Ex: breast trauma, Acute Pancreatic necrosis

→ grossly: opaque white, calcification, chalky white masses

→ Micro: giant cells, dead cells



(J.V.V. imp.)

Table 2.1: Difference between Apoptosis and Necrosis

| | Death of individual cells | Death of group of cells |
|--------------------------|---|---|
| Feature | Apoptosis | Necrosis |
| Definition | Programmed and coordinated cell death | Cell death with degradation of tissues by hydrolytic enzymes |
| Causes | Physiologic and pathologic processes | Pathological, hypoxia, toxins,... |
| Morphology | 1. No inflammatory reaction 2. Death of single cell or small clusters of cells 3. Cell shrinkage 4. Cytoplasmic blebs on membrane 5. Apoptotic bodies 6. Chromatin condensation 7. Phagocytosis of apoptotic bodies by macrophages. | Inflammatory reaction present Death of many adjacent cells Cell swelling initially Membrane disruption Damaged organelles Nuclear disruption Phagocytosis of cell debris by Macrophages |
| Molecular Changes | 1. Lysosomes and other organelles intact 2. Controlled by hormones and enzymes (FAS, BCL-2, p53) <div style="display: flex; justify-content: space-around; font-size: small;"> Stimu. Stimu. Inhib. Stimu. </div> | 1. Lysosomal breakdown with liberation of hydrolytic enzymes 2. Cell death by ATP depletion, membrane damage, free radical injury. |

Cell injury responses تانية وتؤدي إلى Cell injury
 * أسباب الجوع بيجل
 → gangrene
 → intracellular accumulations
 → Pathologic Calcification
 → Cell aging

(7)

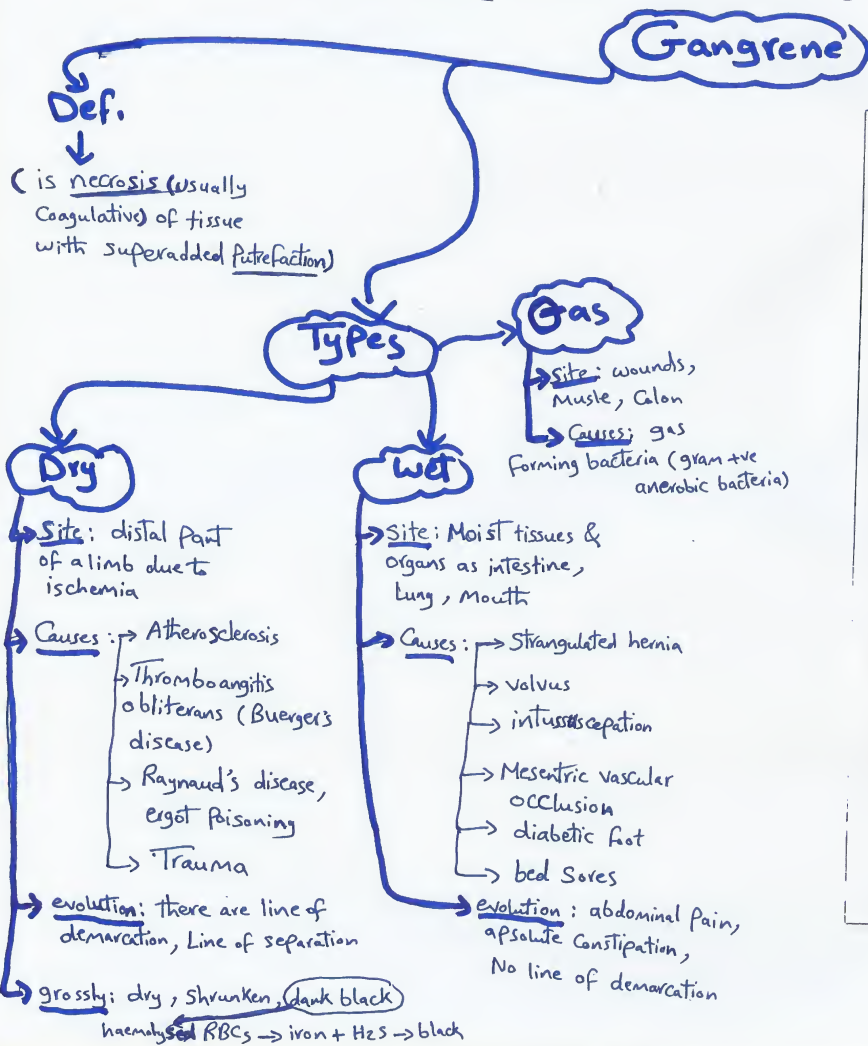


Table 2.2: Difference between of Dry and Wet Gangrene

| Feature | Dry Gangrene | Wet Gangrene |
|---------------------|---|---|
| Site | Commonly limbs | More common in bowel, lung |
| Mechanisms | Arterial occlusion | More commonly venous obstruction, less often arterial occlusion |
| Grossly | Dry, shrunken and black | Moist, soft, swollen, rotten and dark |
| Putrefaction | Limited due to very little blood supply | Marked due to stuffing of organ with blood |
| Line of demarcation | Present at the junction between healthy and gangrenous part | No clear line of demarcation |
| Bacteria | Bacteria fail to survive | Numerous |
| Prognosis | Generally better due to little toxemia | Generally poor due to profound toxemia |

Intracellular accumulations

Def.

→ (accumulation of substances in ~~an~~ abnormal amount can occur within the cytoplasm (especially lysosomes) or the nucleus of the cell)

- if mild → reversible cell injury
- if severe → irreversible cell injury

divided into

increase amount of normal present sub. due to excessive Metabolism
e.g. → Lipids
Proteins
Carbohydrates

increase amount of abnormally present sub. due to abnormal Metabolism
e.g. Lipid storage disease

pigments

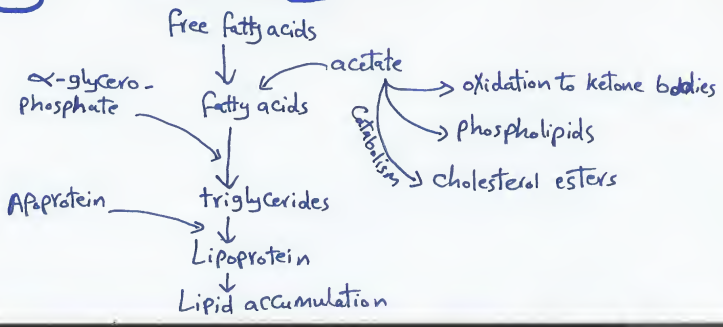
fatty change in liver

→ (accumulation of neutral fat (triglycerides) within cytoplasm of parenchymal cells)

Causes:

- Hyperlipidemia in (obesity, diabetes, congenital)
- alcoholic liver
- starvation in Malnutrition, hypoxia, hepatotoxins.

Pathogenesis (lipid metabolism)



grossly: size: enlarged

Consistency: soft

Edge: rounded

Cut surface: bulging, yellow, greasy

Micro: clear vacuoles (signet ring)

Pigments

→ (Colored substances that are exogenous (from outside body) or endogenous (within body itself))

Endogenous pigments

- Melanin
- Haemoprotein derived pigments (haemosiderin)
- Lipofuscin (wear and tear pigment) (yellowish brown pigment)

Exogenous pigments

- inhaled pigments (Carbon)
- injected pigments (Tattooing)

Pathologic Calcification

Def.

(deposition of Calcium Salts in tissues other than osteoid or enamel is called Pathologic or heterotopic Calcification)

Pathogenesis

Inorganic phosphate binds with Ca ions
↓
Calcium phosphate

Types

Dystrophic

Metastatic

- Normal Ca level (9-11 mg%)
- deposition on abnormal tissue

Causes:

- ① in dead tissue
 - Necrosis (Caseous, Liquefactive, fat, Coagulative)
 - Thrombi
 - Haematomas
 - in breast Cancer
 - Dead Parasites (hydatid cysts and shistosoma eggs)
- ② in degenerated tissues
 - old scars
 - Atheroma
 - Monckeberg's sclerosis in tunica media of muscle.

- increased Ca level
- deposition on normal tissue
- associated with deranged Ca metabolism & hypercalcemia
- Causes:
 - ① excessive mobilization of Ca from bone

- ② Hyperparathyroidism
- bony destructive lesions
- Prolonged immobilization
- ③ excessive absorption of Ca from gut
- Hypervitaminosis D
- Milk alkali syndrome

④ Vitamin D related disorders (vit D intoxication & Sarcoidosis)

| Feature | Dystrophic calcification | Metastatic calcification |
|---------------------|--|--|
| Definition | Deposits of calcium salts in dead and degenerated tissues | Deposits of calcium salts in normal tissues |
| Calcium metabolism | Normal | Deranged |
| Serum calcium level | Normal | Hypercalcemia |
| Causes | -Necrosis (caseous, liquefactive, fat, coagulative) -Thrombi, hematoma. -Dead parasites, old scars -Atheroma. -Monckeberg's sclerosis -Certain tumors | -Hyperparathyroidism -Bony destructive lesions -Prolonged immobilization -Hypervitaminosis D -Milk-alkali syndrome -Sarcoidosis |
| Pathogenesis | Increased binding of phosphates with necrotic and degenerated tissue, which in turn binds to calcium forming calcium phosphate precipitates. | Increased precipitates of calcium phosphate at certain sites due to hypercalcemia e.g. in kidneys, lungs, stomach, arteries, synovium. |

* Choose:

Questions

دى أسئلة
مراجعة متناهي
تراجع على الأسئلة
التالية الى طبيعيا
أثناء المحاضرات

(10)

- 1 Enzymatic fat necrosis occurs in :
 - a) fat embolism b) Tuberculous peritonitis
 - c) Acute haemorrhagic Pancreatitis d) Gas gangrene e) None of the above
- 2 Fragmentation of nuclei in necrotic cells is called
 - a) Pyknosis b) karyolysis c) karyorrhexis d) Autolysis e) None of the above
- 3 Fatty change occurs in :
 - a) Liver b) heart c) kidney d) All of the above e) None of the above
- 4 infarction of the kidney is example of
 - a) Caseating necrosis b) Fibrinoid necrosis c) fat necrosis d) Liquefactive necrosis
 - e) Coagulative necrosis.
- 5 Concerning apoptosis, it's :
 - a) Programmed cell death b) energy dependent process c) No inflammatory Rx
 - d) individual cell necrosis e) all of the above
- 6 Reversible cell injury is characterized by :
 - a) Nuclear changes as pyknosis b) Pathological calcification
 - c) Formation of apoptotic cells d) accumulation of sodium and water inside cell.
 - e) None of the above
- 7 A biopsy from the liver shows that all liver cells are vacuolated and nucleus is giving what is called (signet ring). The cause is :
 - a) Hypoxia , b) Starvation c) Carbon tetrachloride poisoning d) diabetes mellitus e) all the above
- 8 Which of following pathologic changes would be considered irreversible :
 - a) hydropic vacuoles b) karyorrhexis c) fatty change
 - d) Mitochondrial swelling
- 9 Fat can be seen in frozen sections by one of the following stain :
 - a) Methyl violet b) Toluidene blue c) Sudan III
 - d) Congo red
- 10 Caseation necrosis is most characteristic of:
 - a) acute myocardial infarction b) Tuberculosis
 - c) Acute pancreatitis d) brain infarction
 - e) Pulmonary pneumoconiosis
- 11 The following is /are seen in cells undergoing necrosis :
 - a) increase in DNA synthesis b) Fatty change
 - c) Both a & b d) Neither a nor b

* True or False

- ① Hydropic change is an exaggerated form of Cellular Swelling (✓)
- ② Apoptosis is only a physiological process (X)
- ③ Fatty change is abnormal accumulation of Lipoprotein in Parenchymal Cells (X)
- ④ irreversible Cell injury leads to Swelling of Cells due to failure of Na pump (X)
- ⑤ Fatty change is abnormal accumulation of triglycerides in parenchymal Cells (✓)

عشان المراجعة تكون مع حاول تذاكر
الأول أو على الأقل تقرأ المنهج وبعد
كده اسمك المراجعة وتذكر:
(إن الله لا يضيع أجر من أحسن عملاً)

Note

* Cases :-

- ① A 38-year old man has a health screening examination. He has a routine chest X-ray that shows a 2cm nodule in the right lower lobe. The nodule has Focal Calcifications. A wedge resection of the nodule is done - on Microscopic examination the nodule shows Caseous necrosis and Calcification. Which of the following processes explain appearance of Ca deposition?
 a) Dystrophic Calcification b) Apoptosis c) hyperCalcemia d) Metastatic Calcification e) excessive ingestion of Ca
- ② In an experiment, a glass bead is embolized to a branch of renal artery. A day later there's a focal area in which the renal parenchymal cells in the distribution of the occluded artery show karyolysis and karyorrhexis. The outlines of cells are still visible, but the nuclei have lost basophilic staining and the cytoplasm is eosinophilic but pale. What type of necrosis most likely present?
 a) Liquefactive necrosis b) Fat necrosis c) Caseating necrosis d) Coagulative necrosis e) Fibrinoid necrosis

Capsule

تم بحمد الله